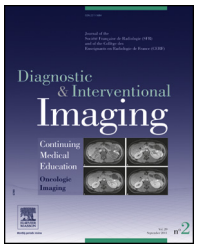
Diagnostic and Interventional Imaging (2014) **95**, 906—916



CONTINUING EDUCATION PROGRAM: FOCUS. . .

Cerebral tumor or pseudotumor?

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KEYWORDS
Brain;
Sarcoidosis;
Histiocytosis;
Behçet;
Tumour

Abstract Pseudotumoral lesions are uncommon but important to identity lesions. They can occur during inflammatory diseases (systemic diseases, vasculitis, demyelinating diseases), infectious, and vascular diseases. Also, in a patient with a treated tumor, pseudo-progression and radionecrosis must be differentiated from the tumoral development. Diagnosis can be difficult on an MRI scan, but some MRI aspects in conventional sequences, diffusion, perfusion and spectroscopy can suggest the pseudotumoral origin of a lesion. Imaging must be interpreted according to the context, the clinic and the biology. The presence of associated intracranial lesions can orientate towards a systemic or infectious disease. A T2 hyposignal lesion suggests granulomatosis or histiocytosis, especially if a meningeal or hypothalamic—pituitary involvement is associated. Non-tumoral lesions are generally not hyperperfused. In the absence of a definitive diagnosis, the evolution of these lesions, whether under treatment or spontaneous, is fundamental.

Any expansive intracranial lesion is not necessarily a tumor. The main diseases that can present a pseudotumoral appearance are systemic diseases, infectious lesions, demyelinating diseases, vascular diseases, vasculitis and radionecrosis. Their diagnosis is crucial since it can lead to an important modification in the way these lesions are managed: no biopsy in the case of vascular or demyelinating lesions, close surveillance or biopsy in emergency for infectious lesions. The diagnosis of these pseudotumoral lesions rests upon imaging, clinical parameters and biology. In neuroradiology, some particular aspects or radiological associations can allude to a pseudotumoral lesion.

The purpose of this article is to present the main pseudotumoral lesions and the way to perform their diagnosis.

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http://dx.doi.org/10.1016/j.diii.2014.08.004

How to make the diagnosis?

Some radiological patterns are typical of the non-tumoral origin of a lesion.

An open-ring enhancement or a restricted demyelinating front are typical of demyelinating lesions.

A gyriform enhancement corresponds to a cortical enhancement, which can be seen in semi-acute stroke, herpes encephalitis and status epilepticus.

A faint brush-like enhancement with well-limited margins without mass effet or T2 WI hyperintensity is typical of capillary telangiectasia.

An eccentric target sign is the hallmark of toxoplasmosis.

When exploring a necrotic lesion, a T2 hypointense rim with surrounding edema is suggestive of an abscess and must lead to perform a diffusion-weighted sequence. A central restricted diffusion confirms the presence of an abscess, which is a diagnosis and therapeutic emergency.

Finally, an edematous bi-thalamic lesion with or without haemorrhage and non-nodular contrast enhancement must suggest a deep venous thrombosis before considering a tumour.

Except for these radiological patterns, the pseudotumoral nature of a lesion can be considered when a lesions presents with atypical features for a brain tumour. Hence, to diagnose a pseudo-tumour, it is necessary to be familiar with the typical aspects of tumoral lesions.

Brain tumours can be single or multiple, and are responsible for mass effect. This mass effect is judged on the deformation of the ventricles and the eradication of sulcis, which must be compared to the size of the lesion. In general, they are in T2 hypersignal. The infiltrating lesions and metastasis are classically in T2 hypersignal with increased ADC, and lymphoma, in T2 hyposignal with decrease of the ADC.

Haemorrhagic lesions can also present with hypo-T2 images and restricted diffusion.

Tumoral lesions, apart from glial lesions with low-grade malignancy, increase in a homogenous way, or partially, with possible necrotic portions [1,2]. Glial lesions with high-grade malignancy and metastasis can appear hyperperfused, since they present with neoangiogenesis, with an increase in rCBV [2]. In spectroscopy, the tumoral infiltration at the periphery of an area of enhancement manifests itself as an increase of the choline peak and a decrease of the NAA peak, and is quite indicative of a glial lesion [2]. In case of mass, a long TE choline/NAA ratio superior to 2 is highly indicative of a tumour [3].

However, if an intra-axial lesion only brings about a small mass effect, a pseudotumoral lesion must be alluded to, particularly a demyelinating disease. The masses in T2 hyposignal, without diffusion hypersignal, support granulomatosis or histiocytosis, especially if a meningeal or hypothalamic—pituitary infection is associated. The cerebral blood volume on the perfusion sequence is not significantly increased in the case of a pseudotumoral lesion, apart from in a few rare cases (see infra: rare cases of neoangiogenesis of inflammatory lesions or injection in strokes). An increase in the choline peak can exist, but in a less higher ratio than in the case of a brain tumour.

The evolution of lesions under medical treatment is obviously fundamental, in particular, the response to steroids being a major argument supporting the inflammatory character of a lesion (apart from the notable exception of primary cerebral lymphoma).

Finally, when suspecting a pseudotumoral of a systemic or infectious origin, it is necessary to carry out an emergency biological etiologic assessment and a CT scan of chest, abdomen and pelvis, in order to conclude as quickly as possible, whether a cerebral biopsy is needed or not.

Main etiologies of pseudotumoral lesions

Inflammatory infections

Systemic diseases

Intra-axial pseudotumors: neurosarcoidosis

Sarcoidosis is a multisystemic granulomatosis, characterised by the presence of epithelioid granulomas, without caseous necrosis [4]. Even if the sarcoidosis lesions are generally extra-axial, actual intra-axial masses, which appear pseudotumoral, can occur. They are most often in T2 hyposignal, and increase consistently, with a perilesional oedema in T2 hypersignal [5—7] (Fig. 1). These masses can be

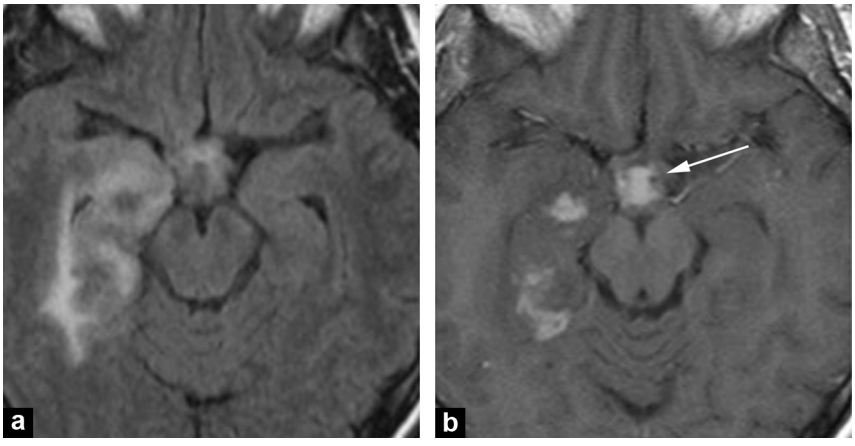

Figure 1. Neurosarcoidosis. FLAIR axial (a) and T1 after injection of gadolinium (b). Parenchymatous lesions, which appear right internal temporal pseudotumoral, in FLAIR hyposignal, enhancing homogeneously after injection and surrounded by oedema in FLAIR hypersignal. Associated hypothalamic infiltration, enhanced after injection.

hyperperfused, and show an increase in the choline peak. A central necrosis is very rare [5]. The main distinct diagnoses are represented by metastases, lymphoma-type primary tumours and pseudotumoral demyelinating lesions.

Neurological sarcoidosis localisations have the characteristic of often being multiple and diffused, affecting the brain, the CNS and cranial nerves at variable degrees. A leptomeningeal increase is frequently associated with these pseudotumoral intra-axial masses, and is considered as a stage prior to the existence of intra-axial granulomas. Indeed, the deep extension of leptomeningeal granulomas along the perivascular spaces can be the start of a parenchymal infiltration [5]. The leptomenigeal infection manifests itself by a micronodular or linear increase in leptomeninges, with a predilection for the suprasellar areas and the base of the skull. An infiltration of the hypothalamic-pituitary axis in T2 hyposignal and increases in cranial nerves can also be associated [8] (Fig. 1). Hydrocephalus is frequent, by meningeal sheathing.

These lesions regress partially or fully under treatment with corticosteroids [9].

Histiocytosis

Different types of histiocytosis are associated with tissue accumulation of histiocytes. Langerhans histiocytosis and non-Langerhans histiocytosis are distinguished. Non-Langerhans histiocytosis include Erdheim—Chester disease, Rosai—Dorfman disease and xanthogranulomas.

Supratentorial intra-axial masses and cerebellar hemispheres can occur during all these types of histiocytosis [10—13]. Most often, they appear in T2 hyposignal and increase homogeneously. Most often, they are in hypersignal in diffusion, related to a T2 effect, the ADC being usually moderately increased. They are responsible for a variable mass effect, according to their size, and are surrounded by an oedema in T2 hypersignal [12]. These masses can be hyperperfused, with a neoangiogenesis in anatomopathology, and show an increase in the choline peak (Fig. 2). They can follow a vascular topography [10].

Pontine lesions can occur during Langerhans histiocytosis, with an irregular contrast enhancement or following a microvascular distribution [10] (Fig. 3).

The main differential diagnoses are metastases and lymphomas. These lesions, difficult to treat, can regress spontaneously or under treatment with corticosteroids and/or chemotherapy [11,14,15], or worsen despite well-guided medical treatments.

Neuro-Behçet's disease

Behçet's disease is a systemic vasculitis. During acute inflammatory phases, the wall of the vessels and the

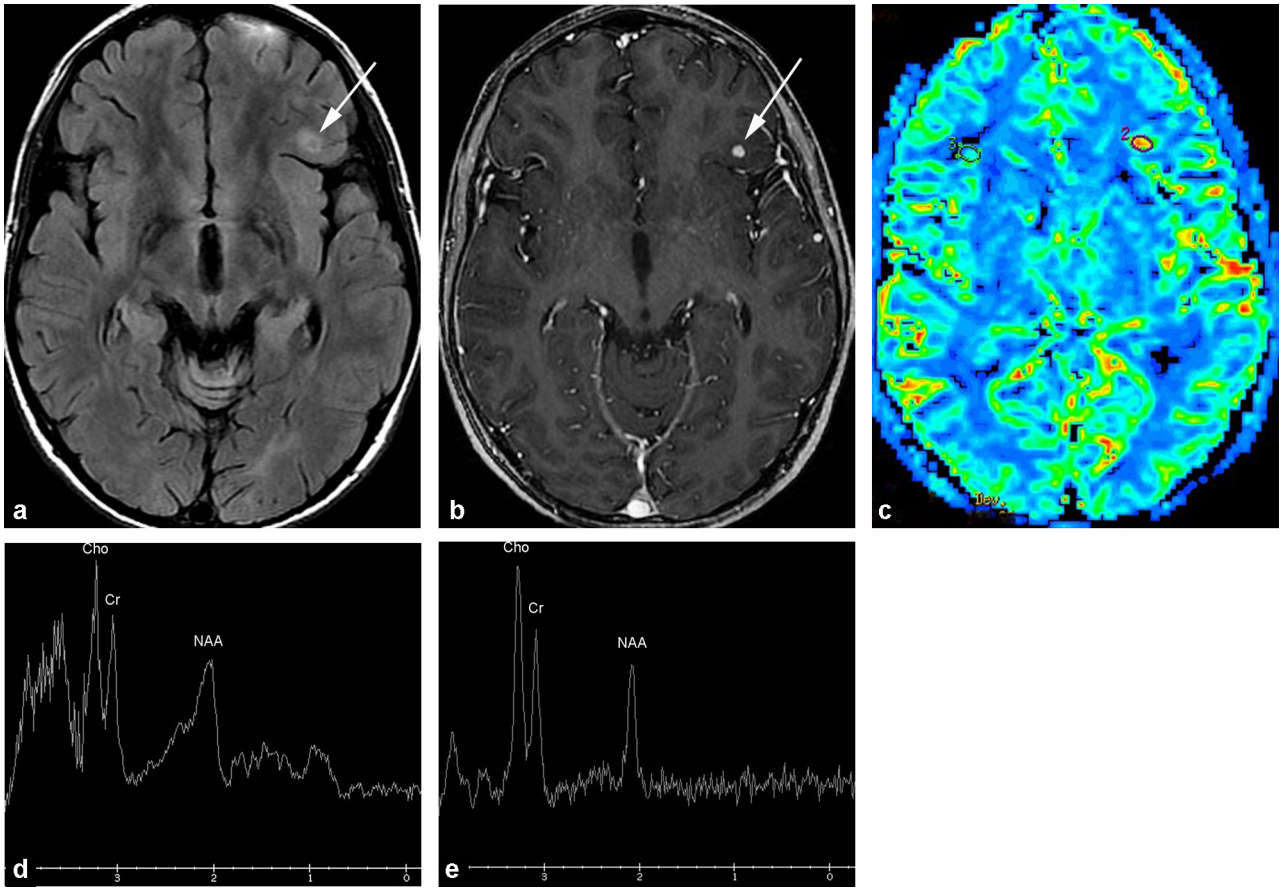

Figure 2. Langerhans histiocytosis. FLAIR axial (a), T1 after injection of gadolinium (b). Perfusion cartography (cerebral blood volume) (c), single voxel spectroscopy at short TE (35 ms) (d) and long TE (135 ms) (e). Sub-cortical left frontal infracentimetric lesion in FLAIR hyposignal, enhancing homogeneously, surrounded by oedema. The lesion is hyperperfused (rCBV at 3.4 times the standard). In the spectroscopy, the Cho/Cr and Cho/NAA connections are increased (> 1), the NAA/Cr connection lowered (< 1).

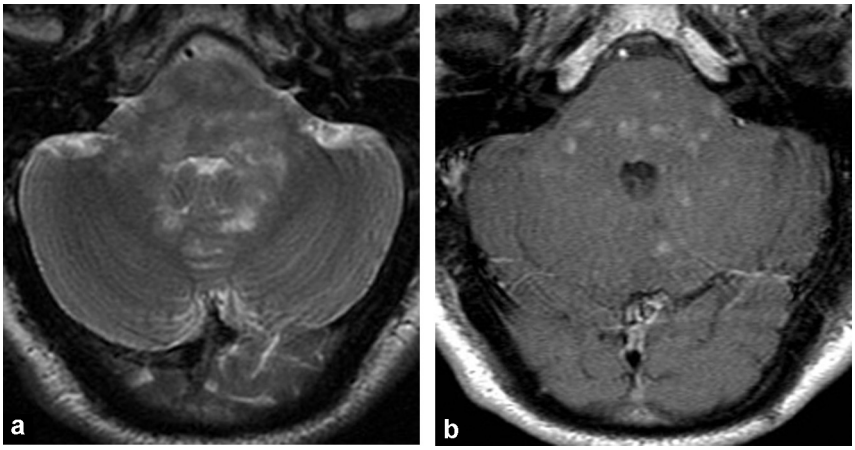

Figure 3. Langerhans histiocytosis. T2 axial (a) and T1 after injection of gadolinium (b). Pontine lesions extended to the middle cerebellar stems and the anterior section of the cerebellar hemispheres, of a heterogeneous signal in T2, with micronodular enhancements. The lesions are surrounded by oedema. The pons is increased in volume.

perivascular cerebral parenchyma are damaged. The oedema and the inflammatory cellular infiltration can be very significant with, in an MRI, areas in T2 hypersignal with a variable size, sometimes confluent in wide ranges, and preferentially touching the pons, the mesencephalon and the basal nuclei, with a possible extension to the internal capsules [6] (Fig. 4). Recent lesions can present with mass effect, contrast enhancement and consequently present a pseudotumoral appearance [16—18]. The main distinct diagnoses are glial lesions, lymphomas or infectious and granulomatous lesions.

In the majority of cases, the size, the mass effect and the enhancement of the acute lesions regress during the first month of treatment, with possibly the complete disappearance of the lesions.

Extra-axial pseudotumors: inflammatory meningeal masses

Pachymeningeal masses can occur during numerous system diseases.

The most frequently etiologies are sarcoidosis and Wegener's granulomatosis. Wegener's granulomatosis is a systemic disease, associating necrotising small vessel vasculitis lesions and giant-cell necrotising and ulcerating extravascular granulomas. The dura mater can be thickened in a diffuse or focal way. These granulomatous lesions, similar to meningiomas, most often appear in T2 hyposignal, and enhance rather peripherally [6—19]. Focal dural masses during Wegener's granulomatosis most often correspond to a transosseous extension of orbital, sinuses and nasal cavities lesions.

Dural, pseudo-meningioma masses, can also be observed during histiocytoses, whether Langerhans or non-Langerhans [6,12,20], Churg—Strauss syndromes [21] or pseudotumoral IgG4 infiltrations [22]. They present with an intense T2 hyposignal and enhance homogeneously after injection.

Intra-ventricular masses in T2 hyposignal and homogeneous enhancement can occur during granulomatoses [23,24] and during histiocytoses. These lesions develop from choroid plexuses [10] or ependymal cells [12,23].

Inflammatory pseudotumors

The main differential diagnosis of lesions occurring during systemic or infectious diseases, whether intra- or

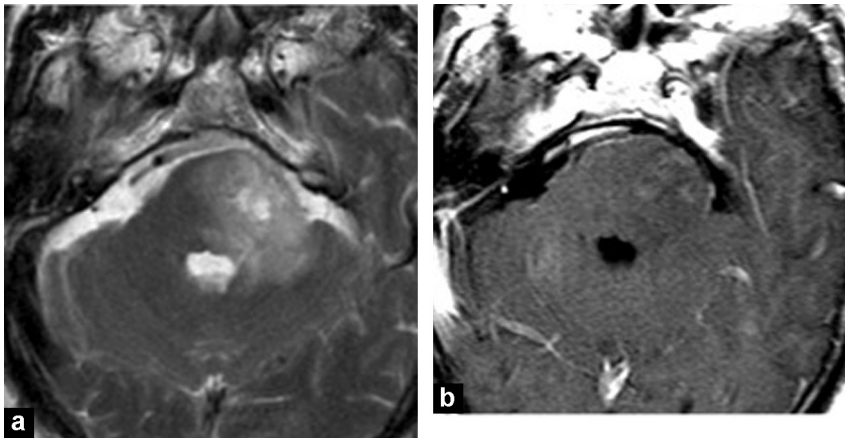

Figure 4. Neuro-Behçet's disease. T2 axial T2 (a) and T1 after injection of gadolinium (b). Pontine lesion extended to the left-middle cerebellar stem in T2 hypersignal, with micronodular enhancement, perilesional oedema with mass effect.

extra-axial, hypointense in T2 and increased intensely, more or less homogeneously, is the ''inflammatory pseudotumor'', without an underlying systemic disease. These masses are characterised by the presence in anatomopathology of a non-specific inflammatory infiltration, and the assessment searching for another localisation being negative. The etiology of these infections is not yet clarified: they could result from an immune response to an infectious agent (EBV, mycobacteria...). These lesions present highly variable responses to medical treatments by corticosteroids and/or immunosuppressants. A surgical resection or treatment by radiotherapy can also be proposed [25—27].

Primary vasculitis of the CNS

Primary vasculitis or idiopathic granulomatous angiitis of the CNS are rare entities of vasculitis, only reaching the CNS and characterised in anatomopathology by a granulomatous inflammatory infection, lymphocystic or necrotising the wall of the leptomeningeal and intra-parenchymatous arterioles [28]. The most frequently reported lesions in an MRI are multiple and bilateral infarctions, intracranial haemorrhages, and more rarely, thickening and enhancements of the artery walls. The MR artery angiogram most often is negative [28].

Parenchymal and leptomeningeal lesions, which appear pseudotumoral, single or multiple, bilateral or only reaching a cerebral hemisphere can be seen in T2 hypersignal, enhance homogenously or heterogeneously with a possible necrotic appearance [29,30]. These pseudotumoral appearances accompany a perilesional oedema and a significant mass effect [31,32] (Fig. 5). The spectroscopy is not specific: the choline peak is barely or not increased, and a lipid peak can be observed [29,31]. The distinct diagnoses are dominated by glial lesions, lymphomas and demyelinating diseases. Development is very variable under treatment with corticosteroids and/or cyclophosphamide-type chemotherapy [29].

Demyelinating disease

Demyelinating lesions can present a pseudotumoral appearance related to a significant inflammatory reaction, with hyperT2 areas appearing extensive, more than 2 cm in diameter. These pseudotumoral forms (Balo's concentric sclerosis, ADEM, Schilder's disease, Marburg virus disease) can mimic a glial tumour, a lymphoma or a metastasis.

Lesions appear in T2 isosignal or hypersignal. Some MRI signs can suggest the diagnosis of demyelinating disease: no or little mass effect compared with the size of the lesion, an incomplete ring enhancement, with in particular, ''C-shape'' enhancement of a sub-cortical lesions [33], a peripheral border with restricted diffusion consistent with a demyelinating front and the presence of a normal vessel within the lesion traducing the venular tropism of the lesions (more visible after injection or in magnetic susceptibility imaging) (Fig. 6). In the case of Balo's concentric sclerosis, a characteristic appearance with multiple concentric areas of demyelination can be seen in T2 and T1 after injection [34]. On spectroscopy, the choline peak is increased and the presence of a lactate peak is usually observed. The perilesional oedema, often barely significant when compared with the size of the lesion, is partially or fully regressive under steroids [33]. However, it is usually necessary to deliver higher corticosteroid doses than in classic acute MS. The duration of enhancement of a demyelinating lesion must not exceed 3 months. Beyond that, it is necessary to allude to a tumoral lesion.

Two very rare entities: CLIPPERS and amyloidoma

CLIPPERS (Chronic Lymphocytic Inflammation with Pontine Perivascular Enhancement Responsive to Steroids) is a rare inflammatory disease of the CNS recently described [35]. In an MRI, lesions are centred on the pons. They appear heterogeneous in T2 hypersignal, present a punctiform and curvilinear enhancement, and can extend to the bulb, to the basal nuclei and to the cerebellar white matter. They do not lead to any significant mass effect. In pathology, lymphocytic

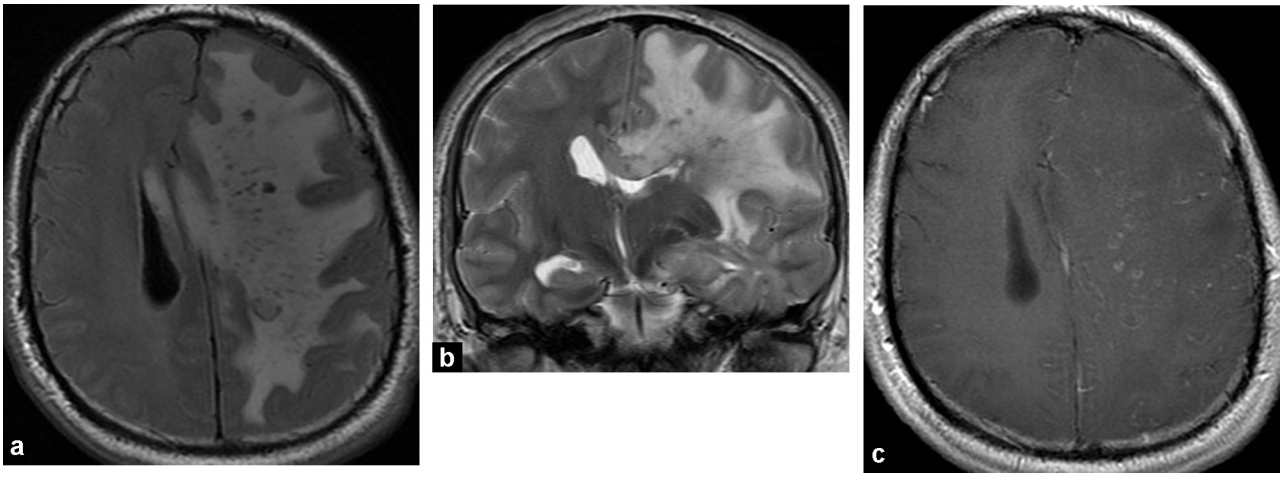

Figure 5. Primary cerebral vasculitis. FLAIR axial (a), T2 coronal (b) and T1 axial after injection of gadolinium (c). Vast range in T2 hypersignal and FLAIR, reaching the left cerebral hemisphere with moderate heterogeneous enhancement and multiple micro bleeding, visible in hyposignal in T2 and FLAIR. Significant perilesional oedema and mass effect with subfalcine and left temporal engagement.

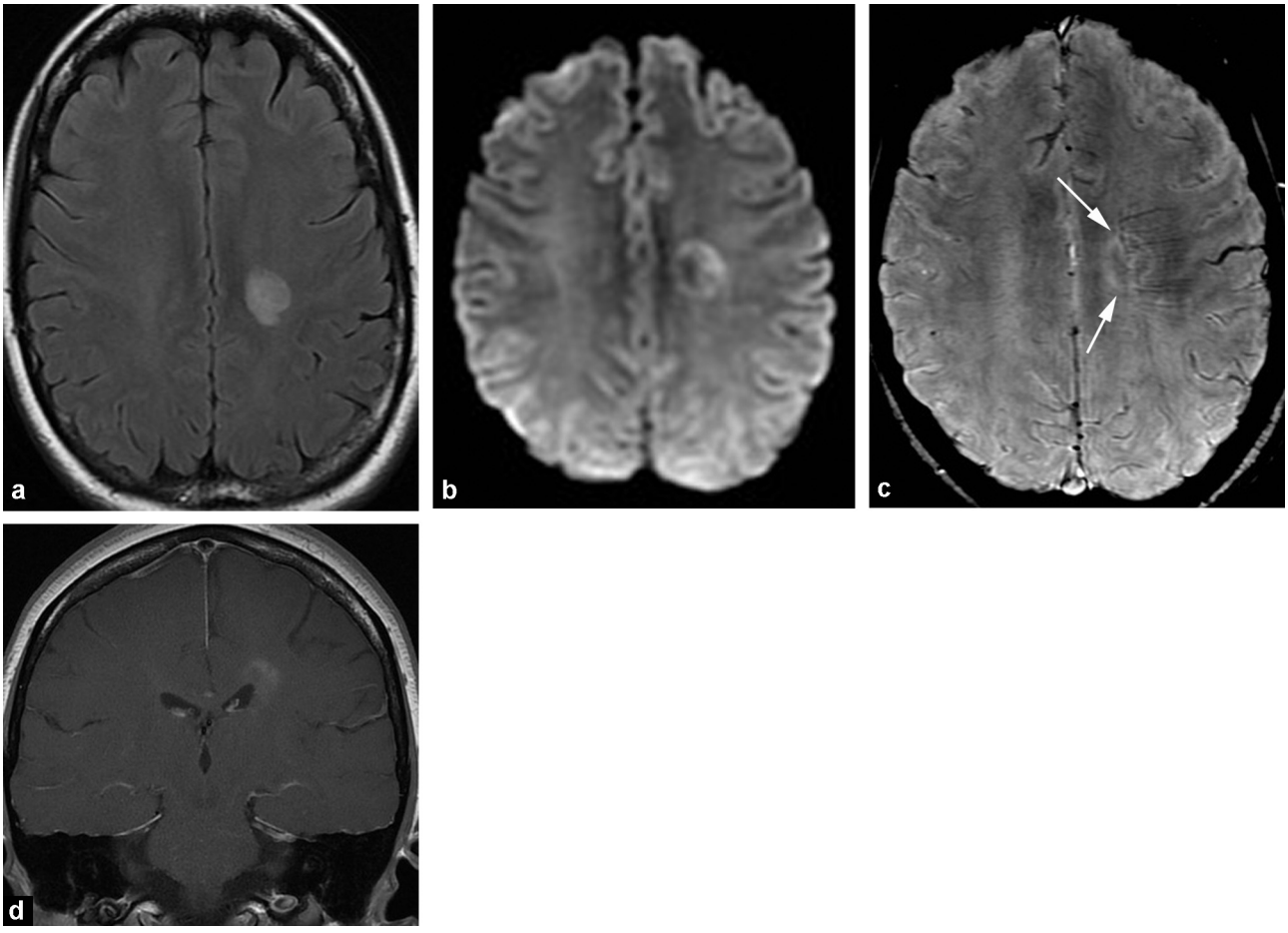

Figure 6. Multiple sclerosis. FLAIR axial (a), diffusion (b), SWI (c), T1 coronal after injection of gadolinium (d). A left juxtaventricular lesion in FLAIR hypersignal, with peripheral border in hypersignal in diffusion, without perilesional oedema, nor mass effect. In magnetic susceptibility imaging (SWI), the lesion appears as if it is being penetrated by multiple veins that appear dilated. After injection, the lesion is enhanced moderately and relatively homogeneously.

infiltration is mainly constituted of T CD3+ lymphocytes and CD20+ lymphocytes.

These lesions are very sensitive to treatments by corticosteroids and/or immunosuppressants [35].

Cerebral amyloidomas are formed from cerebral deposits of amyloid proteins with, in imaging, single or multiple intra-axial masses, developed within the white matter and which can extend to the ventricular walls. Most often, these masses appear in T2 hyposignal, present little or no mass effect and/or perilesional oedema, and their enhancement is variable: discrete or intense [36] (Fig. 7).

Infectious disease

Some infectious diseases can be difficult to distinguish from tumoral lesions, and more particularly, from metastases, because of the very frequent hematogenous dissemination of these two entities, with a preferential localisation of the lesions at the white matter/grey matter junction. Some MRI aspects allow the diagnosis to be steered towards an infectious disease.

Exploring unique or multiple sub-cortical and/or deep lesions, of variable T2 signal and increased peripherically, the presence of an ''eccentric target sign'' (target lesion with an eccentric centre) is highly suggestive of toxoplasmosis. This appearance in imaging is linked to the presence of inflammatory vessels infiltrating to the central section of the lesion [37] (Fig. 8).

Cerebral tuberculomas, as well as some parasites (echinococcosis, brucellosis...) have a variable T2 signal, and can present a homogeneous or heterogeneous ring or cluster enhancement [38].

Perfusion analysis of the wall from these lesions can allow them to be distinguished from glioblastomas or metastases: the rCBV ratio measured between the increased lesion peripheral after injection and the healthy brain tissue is significantly lower in the case of infectious lesions (rCBV < 1) than in cases of high-grade tumoral lesions (rCBV > 5 on average) [39].

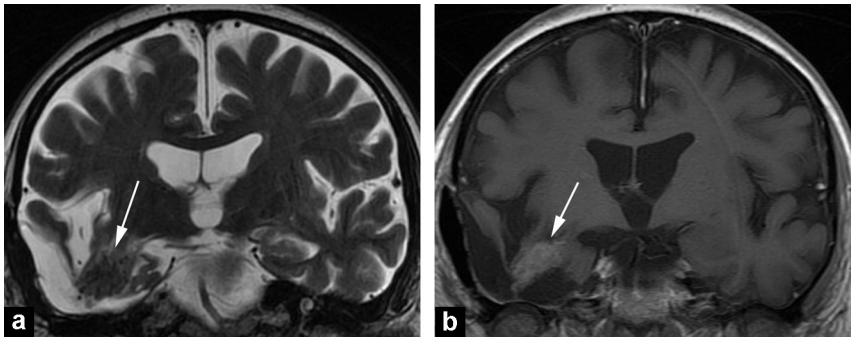

Figure 7. Amyloidoma. T2 coronal T2 (a) and T1 after injection (b). Residual right temporal lesion, in contact with the postoperative cavity, in T2 hyposignal and enhanced moderately and homogeneously, without significant perilesional oedema.

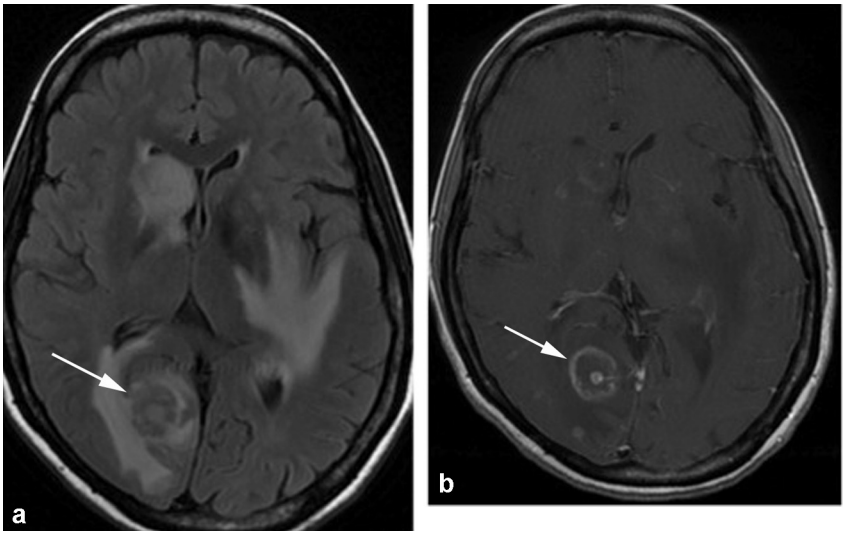

Figure 8. Toxoplasmosis. FLAIR axial (a) and T1 after injection (b). Multiple sub-cortical and deep lesions, enhanced after injection, surrounded by oedema. The most voluminous lesion, right occipital, presents a content in FLAIR hyposignal and a typical target enhancement with an eccentric centre.

Vascular diseases

An ischemic stroke at the sub-acute phase (between day 1 and 2 weeks) appears in T2 hypersignal, present a mass effect with eradication of sulci related to a vasogenic oedema, a gyriform or heterogeneous enhancement relating to the rupture of the blood-brain barrier, which can persist for up to 10 weeks after the stroke, and can present with hyperperfusion known as ''luxury perfusion'', through hyperaemia [40] (Fig. 9). Some ischemic lesions can enhance homogeneously at the sub-acute phase, particularly cerebellar lacunar or basal nuclei infarcts. The appearance on spectroscopy is equally misleading, with a fall in metabolites, sparing of the choline, which therefore appears falsely increased, and the presence of free lipids and lactates (Fig. 9). Consequently, stroke at the sub-acute phase can simulate a tumoral lesion.

Venous infarctions, related to deep vein thrombosis, can also be misdiagnosed as glioma or lymphoma-type tumoral lesions, more particularly when they are unilateral [41]. Indeed, a venous infarction can present itself in an MRI in the form of a unilateral thalamic oedema, more or less extended to the white matter and to the basal nuclei, in T2 hypersignal, associated with possible (micro) bleeding and a discrete and heterogeneous enhancement. Mass effect is common.

Finally, capillary telangiectasias are vascular malformations, constituted of capillaries dilated within a healthy cerebral parenchyma. They can be subtentorial, localised to the pons, or supratentorial. These malformations exert no mass effect, appear in isosignal or in T2 discrete hypersignal, in isosignal in T1 discrete hyposignal, and enhance moderately, and homogeneously after injection. The T2* and SWI sequences allow the diagnosis to be alluded to, with a clear hyposignal component of the malformation over these 2 sequences [42] (Fig. 10).

Tumoral pseudo-progression and radionecrosis

High-grade glial tumours and metastases can be treated by surgery then simultaneous radio- and chemotherapies.

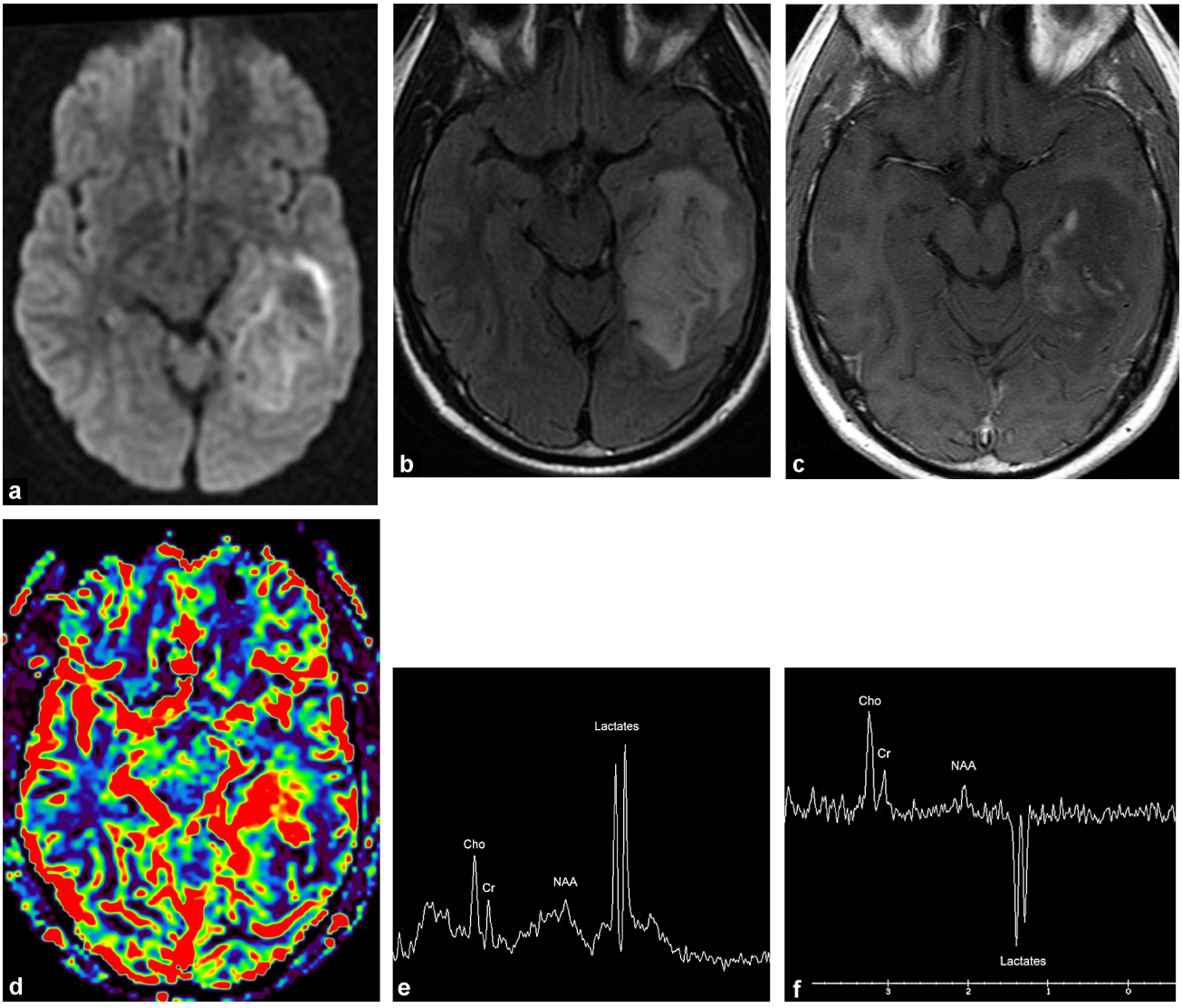

Figure 9. Transient ischemic attack at the sub-acute phase. Axial diffusion (a), FLAIR (b), T1 after injection (c). Cartography of perfusion (cerebral blood volume) (d). Spectroscopy at short TE (35 ms) (e) and long (135 ms) (f). Lesion in the area of the left posterior cerebral artery with a cortical strip in diffusion hypersignal, FLAIR, and with gyriform enhancement and associated vasogenic oedema. The lesion exerts a mass effect with left temporal engagement. It is hyperperfused at 4 times the standard, by hyperaemia. In the spectroscopy, fall in metabolites, apart from choline, which appears falsely increased and presence of free lipids.

The evaluation of the tumoral response is traditionally evaluated according to the MacDonald criteria, based on analysing the volume of lesional enhancement. These criteria present significant limits, as the lesional enhancement can also be the reflection of the disruption of the blood-brain barrier by different mechanisms: post-surgical reorganisation, ischemia, local inflammation to the stopping of radiotherapy with an appearance of pseudo-progression or phenomena of radionecrosis (acute, sub-acute or delayed, in the 18—24 months following the treatment being stopped). During tumoral progression, pseudo-progression and radionecrosis, a perilesional oedema and an increase in the lesional enhancement, sometimes major, can occur. Conventional sequences do not allow the tumoral progression of the pseudo-progression or the radionecrosis to be distinguished with certainty, unlike the sequences of spectroscopy and perfusion, which can demonstrate a NAA decay, a moderate increase in choline with a cho/NAA connection < 1.9 long TE, the presence of a significant peak in lipids/lactates, and an un-increased rCBV in the case of pseudo-progression or radionecrosis [3,43]. In case of doubt, it is advised to re-perform the examination closer (one month), the development of parameters from the spectroscopy and perfusion being more specific than their absolute value.

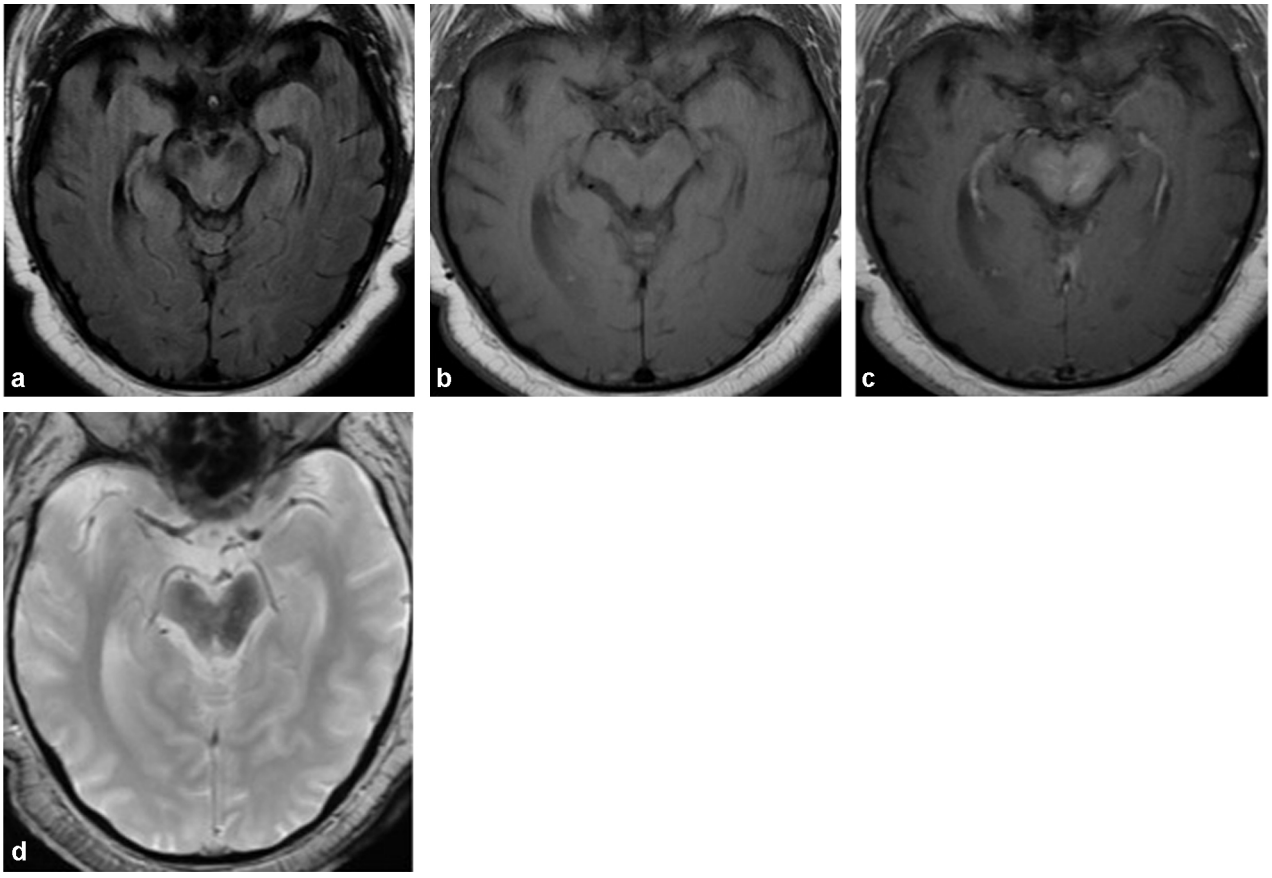

Figure 10. Capillary telangiectasia. FLAIR axial (a), T1 (b), T1 after injection (c), T2* (d). Mesencephalic lesion in FLAIR and T1 isosignal, T2* hyposignal, and enhanced after injection with a striped appearance linked to multiple dilated capillaries. Absence of perilesional oedema and mass effect.

Conclusion

A great variety of inflammatory, infectious and vascular diseases can present themselves in the form of intracranial masses, which appear pseudotumoral. The differential diagnosis between tumor and pseudotumor can therefore can be complex.

TAKE-HOME MESSAGES

- MRI appearances being able to allude to a pseudotumoral lesion:
  - Little or no mass effect;
  - Lesions in T2 hyposignal with normal or increased ADC;
  - 3 particular enhancements: in open-ring (demyelinating disease), gyriform (TIA) and ''eccentric target sign'' (toxoplasmosis);
  - No significant lesional hyperperfusion with a rCBV < 1.75;
  - Choline peak, has not or has barely increased with choline/Cr at long TE < 2.
- Pseudotumoral lesions in T2 hyposignal and enhanced:
  - Granulomatosis, histiocytosis, Behçet's Disease, Churg—Strauss Syndrome, IgG4 infiltration;
  - Inflammatory pseudotumor;
  - Amyloidoma;
  - Tuberculoma.
- Pseudotumoral lesions of the brain stem:
  - Langerhans histiocytosis;
  - Neuro-Behçet's disease;
  - CLIPPERS;
  - Demyelinating lesion;
  - Capillary telangiectasia.

Clinical case

Female of 49 years old with medical history of diabetes insipidus and infertility. Loss of consciousness (Fig. 11).

Questions

1. How would you describe the cerebral lesions?

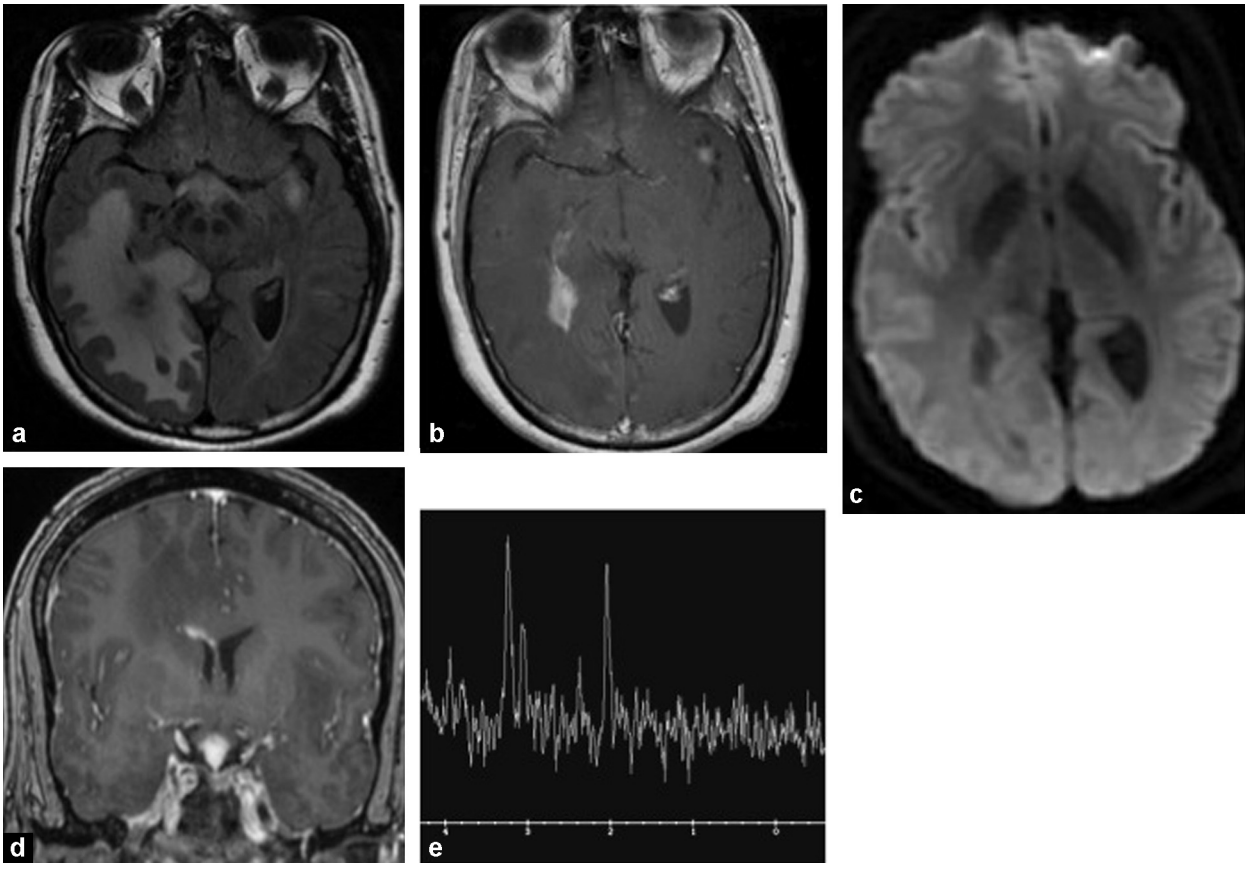

Figure 11. FLAIR axial (a), T1 after injection (b), diffusion (c), T1 coronal after injection (d), single voxel spectroscopy at long TE (135 ms) centred on the juxtaventricular lesion of the right ventricular intersection (e).

2. What are the three main diagnoses to allude to when faced with this type of lesional association?
3. In this case, which MRI appearances must a pseudotumoral lesion rather be alluded to?

Answers

1. The MRI highlights several intracranial lesions:
   - a juxtaventricular lesion in the area of the right ventricular intersection, in FLAIR hyposignal and diffusion, and enhanced homogeneously after injection. This lesion is surrounded by a significant oedema in FLAIR hypersignal. On spectroscopy, the choline/Cr and cho/NAA ratios are increased. No lipid peak can be seen;
   - a juxtaventricular lesion, enhanced after injection on contact with the anterior horn of the right lateral ventricle;
   - a sub-cortical left insular nodular lesion, enhanced after injection and surrounded by an oedema in FLAIR hypersignal;
   - a diffused and nodular thickening of the pituitary stem, enhanced after injection.
2. Facing juxtaventricular sub-cortical and pituitary stem lesions, neurosarcoidosis, histiocytosis and a primary cerebral lymphoma can be alluded to.
3. The lesion in the area of the right ventricular intersection is in FLAIR hyposignal and diffusion. A lymphomatous localisation should appear in FLAIR hyposignal and diffusion hypersignal. In the spectroscopy, no lipid peak, characteristic of a lymphomatous localisation is seen.

Diagnosis

Neurosarcoidosis.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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